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Clinical Study

Dysfunctional sleep beliefs in Parkinson's disease: Relationships with subjective and objective sleep

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ABSTRACT

Disturbed sleep is common in Parkinson's disease and has a detrimental impact on functioning and quality of life. While the progression of the disease contributes to the aetiology of sleep problems in Parkinson's disease, it is unknown whether an individual's beliefs and attitudes about sleep play a role. In this study we sought to investigate whether dysfunctional beliefs and attitudes about sleep could be related to subjective and objective measures of sleep disturbance in Parkinson's disease. Ninety-three patients with Parkinson's disease completed the Dysfunctional Beliefs and Attitudes about Sleep 16 item questionnaire, which comprises four domains: Expectations, Worry/Helplessness, Consequences and Medication. Patients also completed the Pittsburgh Sleep Quality Index questionnaire and Beck Depression Inventory-II. Patients wore actigraphy watches and completed sleep diaries for 2 consecutive weeks, recording measures of sleep disturbance including Sleep Onset and Offset, Wake After Sleep Onset, Sleep Efficiency, and Wake Bouts per hour. Greater dysfunctional beliefs and attitudes in the domains of Worry/Helplessness and Medication were associated with lower perceived sleep quality and greater depressive symptoms. However, no relationships were found between dysfunctional beliefs and attitudes about sleep and any objective actigraphic measure of sleep disturbance. These findings suggest that beliefs and attitudes about sleep in Parkinson's disease are associated with mood disturbance, rather than objective measures of sleep. Thus it is possible that interventions targeting mood may lead to more accurate perceptions of sleep and improved quality of life in Parkinson's disease patients.

1. Introduction

Disturbed sleep is a frequent complaint in many clinical populations and is linked to a range of physical and neuropsychiatric symptoms. Despite being primarily known as a movement disorder, sleep disturbances are being increasingly recognised as an integral feature of Parkinson's disease (PD) [1,2]. Over two thirds of patients are symptomatic [3] with a variety of sleep disorders reported including excessive daytime sleepiness, insomnia, sleep maintenance problems and rapid eye movement sleep behaviour disorder [2–4]. Significantly, sleep problems in PD have been previously linked with physical function, quality of life, depression, psychosis, and an increased risk of developing dementia [3,5,6]. Furthermore, such disturbances also impact upon spousal carer sleep, as well as their perceived level of burden, often providing an impetus for nursing home placement [7,8].

In other clinical populations, research has shown that sleep expectations and being "overly worried" about sleep are factors which may exacerbate sleep disturbance [9]. In turn this heightened worry causes autonomic arousal and emotional distress. This leads to selective attention where an individual can focus on the deficit and overestimate it. Previous studies in non-PD cohorts (such as primary insomnia, late life insomnia, menopause, major depressive disorder and other mood disorders) have demonstrated that individuals with dysfunctional beliefs and attitudes about sleep (DBAS) feel that their health will suffer if they do not attain a minimum number of hours sleeping per night [10–13]. These patient groups are prone to developing a form of "performance anxiety" [14] and may adopt strategies to compensate for this perceived lack of sleep [9]. Such strategies include excessive napping, staying in bed for longer periods or utilising agents such as caffeine or other drugs, all of which may disrupt their sleep–wake patterns further.

The Dysfunctional Beliefs and Attitudes about Sleep 16 item Questionnaire (DBAS-16), is a validated scale that can measure

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these perceptions [15]. Higher ratings on the DBAS-16 tend to be associated with poorer health and wellbeing [9,15] and are correlated with greater levels of sleep disturbance [12,13]. Thus a greater appreciation of these factors may help direct targeted sleep–wake psychological therapies amongst clinical populations.

To our knowledge no work has evaluated DBAS using the DBAS-16 in a sample of PD patients. Therefore, this study was undertaken to identify the level of DBAS in PD and determine whether these perceptions of sleep could be correlated with self-reported sleep disturbance, as measured by questionnaires and, more importantly, with objective measures of sleep disturbance as recorded by actigraphic assessment.

2. Methods

2.1. Recruitment

Ninety-three patients with PD (mean age 63.9 years ± standard deviation [SD] of 7.9 years, 61.3% male) were recruited from the Parkinson's Disease Research Clinic at the Brain and Mind Research Institute, University of Sydney (Table 1). Exclusion criteria were a history of stroke, neurological disorder other than PD, diagnosis of obstructive sleep apnoea, head injury with loss of consciousness $\geqslant$30 minutes, medical conditions known to affect cognition (such as cancer), psychiatric illness, shift work, transmeridian travel within the prior 60 days, and use of medications (other than those for PD) known to affect sleep and/or melatonin secretion including beta-blockers, lithium or benzodiazepines. All patients satisfied the United Kingdom Parkinson's Disease Society Brain Bank criteria [16] and 71% of patients were on dopaminergic medication. Of those on dopaminergic medication there was an average L-dopa dose equivalence of 700 mg (± SD 481 mg). In addition, 22% were taking antidepressant medications. No patients taking hypnotics were included in the study.

Patients were assessed on the Unified Parkinson's Disease Rating Scale – motor symptoms (UPDRS-III) [17] and were staged according to the Hoehn and Yahr (H&Y) scale [18]. None of the patients were deemed as having dementia according to Movement Disorder Society Task Force criteria [19] or major depression by the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition [20] following consensus rating by a neurologist and a neuropsychologist. Mood was further assessed using the Beck Depression Inventory (BDI-II) [21].

Permission for the study was obtained from the University of Sydney research ethics committee and all participants provided written informed consent.

Table 1
Demographic, clinical, self-report, Dysfunctional Beliefs and Attitudes about Sleep 16 item Questionnaire and actigraphy data for patients with Parkinson's disease

	Mean	Standard deviation	n
Demographic data			
Disease duration (years)	5.6	5.1	90
Age (years)	63.9	7.9	93
L-Dopa dose equivalent (mg)	700	481	65
Clinical data			
UPDRS-III	23.0	10.1	93
H&Y	1.9	0.7	92
Self-report data			
PSQI	6.4	3.3	91
BDI-II	9.8	6.1	92
DBAS-16 data			
Total DBAS-16	65.7	24.3	93
Expectations	11.1	4.4	93
Worry/Helplessness	23.9	10.6	93
Consequences	21.2	9.2	93
Medication	9.6	6.2	93
Actigraphy data			
Sleep Onset (hh:mm)	22:42	01:13	93
Sleep Offset (hh:mm)	07:02	01:02	93
WASO (minutes)	51.9	32.1	93
Sleep Efficiency (%)	89.6	6.4	93
Wake Bouts per hour	5.0	2.6	93

BDI-II = Beck Depression Inventory, DBAS-16 = Dysfunctional Beliefs and Attitudes about Sleep 16 item Questionnaire, hh:mm = hours:minutes in 24 hour time, H&Y = Hoehn and Yahr scale, PSQI = Pittsburgh Sleep Quality Index, UPDRS-III = Unified Parkinson's Disease Rating Scale – motor symptoms, WASO = Wake After Sleep Onset.

3. Measures

3.1. DBAS-16

The DBAS-16 asks patients to rate how much they agree with a series of statements relating to sleep [15]. Responses are made using an 11 point Likert scale (0 = strongly disagree, 10 = strongly agree). The more dysfunctional the beliefs of an individual, the higher they will rank each statement resulting in a greater total score.

The four domains of the DBAS-16 are (1) Expectations: for example, I need 8 hours of sleep to feel refreshed and function well the next day; (2) Worry/Helplessness: for example, I am worried that I may lose control over my abilities to sleep; (3) Consequences: for example, I avoid or cancel obligations after a poor night's sleep; and (4) Medication: for example, medication is probably the only solution to sleeplessness.

3.2. Pittsburgh Sleep Quality Index

To evaluate self perceived sleep quality, patients completed the Pittsburgh Sleep Quality Index (PSQI). This is a 19 item questionnaire that creates seven component scores equally weighted from 0–3. The sum of the component scores generates a global score between 0 and 21; therefore, the higher the global score, the worse the sleep quality. Those with a PSQI score more than or equal to 5 are classed as having sleep disturbance [22]. The PSQI has previously been used to assess sleep quality in PD patients [23].

3.3. Actigraphy

Actigraphy is a frequently used and well validated objective measure of sleep–wake behaviour in non-PD [24,25] and PD samples [26,27]. Actigraphy was collected for 14 days according to an established protocol [26] using wrist actigraphy watches (MiniMitter Actiwatch Spectrum; Minimitter-Respironics Inc., Bend, OR, USA) that measure movement and intensity of light exposure. As described previously, patients were instructed to wear the watch on the wrist less severely affected by PD and complete a daily sleep diary [26]. The data were downloaded and scored using Actiware 5.0 software with Actiwatch Firmware Version 01.01.0007 (Minimitter-Respironics Inc.). While it is noted that actigraphy can only identify "rest" intervals where the patient is not moving, the term "sleep" has been used here for ease of interpretation. Manual scoring corrections were applied by a trained technician who incorporated information recorded in the sleep diaries. Naps were excluded so that only one sleep interval was scored for each 24 hour window. The wake threshold value (i.e., the number of activity counts used to define wake) was set to medium sensitivity of 40.0 activity counts per 30 second epoch.

Outcome variables from the actigraphy assessment used in analyses included:

(1) Sleep Onset Time (hh:mm): the average time at which sleep in bed commenced

(2) Sleep Offset Time (hh:mm): the average time at which sleep in bed ceased
(3) Wake After Sleep Onset (WASO) (minutes): the mean total time awake after sleep onset
(4) Sleep Efficiency (%): the mean percentage of time in bed that was sleep
(5) Wake Bouts per hour: the mean number of disturbances per hour spent in bed.

3.4. Statistical analysis

All analyses were performed using the Statistical Package for the Social Sciences version 20 (SPSS Inc., Chicago, IL, USA). Kolmogorov–Smirnov tests were used to assess data distribution. Correlations were used to examine relationships between demographic variables and actigraphy measures with the DBAS-16; Pearson's correlations were used on normally distributed variables and Spearman's correlations were used on variables that were not normally distributed. Partial correlations were constructed based on significant correlations to remove possible confounding relationships. Due to the fact that PSQI and the Medication domain of the DBAS-16 were non-parametric variables, it was not possible to run standard partial correlations to evaluate the relative contribution of confounding variables. However, it was possible to remove one confound using the following formula,

$$r_{x.y.z} = \frac{r_{xy} - r_{xz}r_{yz}}{\sqrt{1 - r_{xy}^2}\sqrt{1 - r_{yz}^2}}$$

where x was the dependent variable, y was the independent variable and z was the potential confound [28]. All analyses used an alpha of 0.05 and were two-tailed.

4. Results

Patients had an average disease duration of 5.6 years (± SD 5.1 years), a UPDRS-III of 23.0 (± SD 10.1) and H&Y score of 1.9 (± SD 0.7). No patients reported significant major depression and on average had minimal depressive symptoms (BDI-II = 9.8 ± SD 6.1) (Table 1).

Level of depression as measured by BDI-II correlated with the Total DBAS-16 (r = 0.272, p = 0.009) and the domains measuring Worry/Helplessness (r = 0.286, p = 0.006) and Medication (r_s = 0.295, p = 0.004). Age, motor symptom severity, L-dopa dose equivalent and disease duration were not associated with Total DBAS-16 or any of its domains.

L-dopa dose equivalent was negatively correlated with Sleep Efficiency (r = −0.398, p = −0.001) and positively correlated with WASO (r = 0.412, p = 0.001).

4.1. Relationship between DBAS-16 and subjective sleep quality (PSQI)

Poorer self-rated sleep quality, as measured by the PSQI, was associated with greater dysfunctional beliefs about sleep (Total DBAS-16, r_s = 0.272, p = 0.009). Specifically, PSQI scores appeared to relate most strongly to the Worry/Helplessness domain (r_s = 0.286, p = 0.006), and the Medication domain (r_s = 0.295, p = 0.004) but not to the sleep Expectations or Consequences domains.

Since DBAS-16 scores were also associated with depressive symptoms, these analyses were repeated after controlling for BDI-II scores. Results showed that the relationship between PSQI and Total DBAS-16 score (partial r = 0.226, p = 0.029) and Worry/Helplessness (partial r = 0.211, p = 0.042) remained significant. However, the relationship between PSQI and the Medication domain was no longer significant (partial r = 0.159).

The relationship between DBAS-16 profile and sleep quality was also examined by comparing those with and without self-reported sleep disturbance as classified by a cut-off score of 5 on the PSQI. Accordingly, those with sleep disturbance (PSQI $\geqslant$ 5, n = 60) had greater dysfunctional beliefs than those without (PSQI <5) for the Total DBAS-16 (t = 2.142, p = 0.035) as well as the domains of Worry/Helplessness (t = 2.153, p = 0.034) and Medication (t = 3.356, p = 0.001). However the domains regarding Expectations and Consequences were not significantly different across the two groups.

4.2. DBAS-16 scores versus objective sleep

In order to determine whether dysfunctional beliefs about sleep were associated with actual sleep disturbance, DBAS scores were correlated with objective measures of sleep as recorded by actigraphy. There were no significant correlations found between Total DBAS-16 or any DBAS-16 domain score and WASO, Sleep Efficiency or Wake Bouts per hour. The only significant correlation between sleep beliefs and actigraphic measures was between the Expectations domain of the DBAS-16 and Sleep Offset Time (r = 0.224, p = 0.031). No other correlations were found for Sleep Onset or Offset Times with the DBAS-16.

5. Discussion

This study shows that in patients with PD, DBAS relate to a patient's perception of sleep quality as measured by the PSQI. Specifically, heightened worry and concern about sleep was found to relate to poorer perceived sleep quality. This relationship may be bidirectional and thus it is unclear if this association represents an overestimation of a sleep deficit (invoked by dysfunctional beliefs) that in turn creates unrealistic goals (that patients fail to meet) creating a perception of sleep disturbance. Alternatively, this relationship may reflect misplaced blame created by patients attributing disease symptoms to their perceived sleep disturbance therefore altering the perceived effect of sleep disturbances and changing beliefs.

In contrast, the DBAS-16 did not relate to actual sleep disturbance as measured objectively by actigraphy, aside from Sleep Offset time, which correlated with the domain recording Expectations. Whilst this study found no evidence that DBAS are associated with sleep disturbance as measured by actigraphy, it is possible that this objective measure of sleep is insensitive to some forms of sleep disorder including unrefreshing sleep, hypersomnolence, bruxism, hypopnea, nightmares or excessive snoring, some of which can be relevant in PD. In addition, it is also possible that symptoms such as restless leg syndrome that cannot easily be identified using actigraphy may be related to a patient's beliefs and attitudes about sleep. Future studies to address these possibilities would need to include formal polysomnography.

Interestingly, only the Total DBAS-16 score and the domain exploring Worry/Helplessness withstood correction for a patient's current levels of depression. This suggests that although depression is a contributor to dysfunctional beliefs and attitudes about sleep in PD there is likely to be a more pervasive deficit. It is logical that Worry/Helplessness about sleep is of particular relevance in PD as sleep disruption is common and can be a distressing symptom of the disease. Therefore it follows that patients will be worried about losing control of their sleep, particularly when this has negative consequences. This elevated worry may further exacerbate both perceived and objective measures of sleep disturbance. This would also explain the correlation between expectations and Sleep Offset time as this observation may suggest that those with higher expectations of sleep stay in bed longer attempting to correct the perceived deficit. Not having a stronger correlation

between the DBAS-16 and Sleep Onset time shows that patients had not adopted the altered behaviour of going to bed earlier to compensate.

Symptoms such as executive dysfunction and loss of attention may be less prominent symptoms to a patient with a well practiced daily routine but sleep disturbance could be more obvious, potentially causing alarm and creating worry. The progressive and relentless nature of a neurodegenerative disease like PD may also create a "learned helplessness" within a population that is more likely to manifest symptoms that are most frequent and evident within a daily activity.

The interpretations of our findings for the Medication domain of the DBAS-16 are likely to represent a more complex relationship mediated by other factors. The loss of a significant relationship between perceived sleep quality and the Medication domain of the DBAS-16 after controlling for depressive symptoms suggests that those patients who are more susceptible to depression are more likely to have a dependence on medication and are therefore more likely to hold dysfunctional beliefs. Furthermore, it is well recognised that using regular hypnotics can create a reliance on these medications, which may be detrimental to health [29]. Although individuals taking hypnotics were excluded from recruitment in this study, many patients are reliant on their PD medication to reduce physical discomfort. As such, it is possible that such "dependence" could promote dysfunctional beliefs. Therefore PD patients may develop the perception that tablets represent a vital approach to avoiding sleeplessness, perhaps operating via the reduction of physical symptoms, and consequently patients could overestimate the level of sleep disturbance they experience.

The Expectations and the Consequences domain of the DBAS-16 were not significantly correlated with any of the subjective, objective or phenotypic measures recorded in this study. This lack of correlation around Expectations may be due to a "desensitisation" with patients having learnt to expect poor sleep with disturbances. There may also be an acceptance by PD patients that the consequences of poor sleep, such as tiredness, lack of energy, loss of function, irritability, depression and anxiety, simply represent another feature of their condition.

Cognitive behavioral therapy (CBT) programs have been shown to be effective in reducing DBAS, improving sleep and quality of life in other clinical populations with sleep disturbances [30]. Therefore, the development of CBT programs or other sleep and mood focused psychoeducation programs for use in PD may represent one practical approach to the management of this problem. The results of the current study would suggest that such programs would specifically need to incorporate cognitive restructuring of feelings of worry and beliefs about medication, as well as improving affective symptoms. Reducing DBAS could potentially improve mood and perceived sleep efficiency leading to a better quality of life and fewer nursing home placements. Such a strategy would also need to recognise the fact that despite optimal management, patients with PD are still affected by troublesome symptoms including their physical parkinsonism, pain, nocturia and sleep disturbances.

It is well recognised that PD is a heterogeneous disease comprised of subgroups including younger onset, tremor and non-tremor dominant populations [31] and therefore future work may need to explore the influence of such phenotypes on the pattern of dysfunctional beliefs observed in PD. Similarly, the patients included in this study did not represent the full spectrum of clinical disease severity and it is possible that DBAS-16 profiles may be different in patients who have more advanced PD. Longitudinal study of patients may help delineate the directionality of the relationship between DBAS and the perceived sleep efficiency. It is hoped that challenging dysfunctional beliefs about sleep in PD, such that patients could more readily cope with their sleep difficulties, may reduce the overall burden of disease for both the patient and their carers.

Conflicts of interest/disclosures

The authors declare that they have no financial or other conflicts of interest in relation to this research and its publication.

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